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Research Progress on Acute Kidney Injury in Patients with Cardiogenic Shock Undergoing Venoarterial Extracorporeal Membrane Oxygenation

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Abstract: Cardiogenic shock (CS) is a life-threatening syndrome characterized by peripheral hypoperfusion and organ dysfunction caused by primary heart disease. Venoarterial extracorporeal membrane oxygenation (VA-ECMO) is a temporary mechanical circulatory support device for CS, used in cases of profound shock, biventricular failure, respiratory failure, and cardiac arrest that require urgent maximal support. While VA-ECMO provides effective tissue perfusion and ensures oxygen supply to organs, it is also associated with severe complications, among which acute kidney injury (AKI) is one of the most common and serious. To date, no comprehensive review has been conducted on the pathophysiology, influencing factors, and treatment of AKI associated with VA-ECMO. This paper aims to elaborate on the pathophysiological mechanisms, influencing factors, and treatment options for AKI in patients with CS undergoing VA-ECMO, providing clinical and nursing references.

Keywords: Cardiogenic shock; Venoarterial extracorporeal membrane oxygenation; Acute kidney injury; Review

Online publication:

1. Overview of cardiogenic shock

Cardiogenic shock (CS) is a life-threatening condition characterized by acute end-organ hypoperfusion, which, if not promptly reversed, can rapidly lead to multi-organ failure and even death. CS encompasses a range of clinical syndromes with various etiologies, making it crucial to identify the cause in clinical practice. Acute myocardial infarction, acute decompensated heart failure, left ventricular outflow tract obstruction, right ventricular pump failure, acute valvular regurgitation, cardiac rupture, and post-pericardiotomy syndrome can all trigger CS, with acute myocardial infarction being the leading cause. The diagnostic criteria for CS include systolic blood pressure < 90 mmHg, urine output < 30 mL/h, lactate > 2 mmol/L, mixed venous oxygen

saturation < 60%, altered consciousness for 6 hours, and unresponsiveness to optimal treatment plans [1].

Current treatment modalities for CS include pharmacological and non-pharmacological interventions. Pharmacological treatments mainly involve vasoconstrictors and inotropic agents, while the efficacy of vasodilators remains contentious. Commonly used vasopressors and inotropic agents include dopamine (moderate to high doses), dobutamine, norepinephrine, epinephrine, milrinone, and levosimendan. Non-pharmacological treatments primarily consist of mechanical support and heart transplantation. Mechanical support includes intra-aortic balloon pump (IABP), extracorporeal membrane oxygenation (ECMO), and mechanical ventilation. For patients whose hemodynamic instability cannot be resolved by the aforementioned methods, heart transplantation may be considered.

With the latest advancements in mechanical circulatory support (MCS) technology, the management of CS has evolved. Current consensus guidelines advocate for the early deployment of MCS in refractory CS. Among the available temporary MCS devices, VA-ECMO holds distinct advantages, offering rapid bedside deployment and providing biventricular and pulmonary support, thereby preventing end-organ failure due to prolonged tissue hypoperfusion [2]. Timely ECMO implantation is an effective strategy for reducing the duration of end-organ ischemia, which is critical for the management of this patient population [3].

2. Current status of extracorporeal membrane oxygenation use

Extracorporeal membrane oxygenation (ECMO) is a life-saving treatment for patients with severe respiratory and/or cardiovascular failure [4]. ECMO can be divided into two types based on the mode of blood return: (1) Venoarterial ECMO (VA-ECMO) for patients with refractory cardiogenic shock or combined cardiopulmonary failure, and (2) Venovenous ECMO (VV-ECMO) for patients with potentially reversible causes of respiratory failure. VA-ECMO is used to treat refractory cardiogenic shock (CS) caused by conditions such as acute myocardial infarction, electrical storm, myocarditis, and pulmonary embolism. It serves as a bridge to recovery of native heart function, ventricular assist devices, or heart transplantation. Over the past decade, ECMO usage has significantly increased in intensive care units, emergency departments, inter-hospital transfers, operating rooms, and during cardiopulmonary resuscitation. As of June 2023, a total of 202,449 patients worldwide have received extracorporeal life support treatment [5]. ECMO has transitioned from an experimental phase into widespread use, with rapid increases in the number of cases, and has now entered a period of systematic management. Meanwhile, the management of complications related to ECMO has garnered increasing attention.

3. Status of acute kidney injury during VA-ECMO use

Acute kidney injury (AKI) is a clinical syndrome characterized by acute functional impairment and kidney damage. It is relatively common among hospitalized patients in the United States, with approximately 2.2 million cases per year [6]. According to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines, AKI is defined by an increase in serum creatinine ≥ 0.3 mg/dL (≥ 26.5 μmol/L) within 48 hours, or an increase in serum creatinine to 1.5 times or more of the baseline within the past 7 days, or a urine output ≤ 0.5 mL/kg/h for 6 hours. Meeting any of these criteria qualifies as AKI. It is frequently seen in CS and is closely associated with increased patient mortality [7]. AKI is the most common and serious complication during ECMO use, significantly affecting patient outcomes [8,9]. Literature indicates that the occurrence of AKI is associated with

the type of ECMO support. Compared to VV-ECMO patients, VA-ECMO patients have a higher probability of developing AKI [10]. However, some studies have found no statistically significant difference in the risk of AKI or severe AKI between patients using different ECMO modes. Nonetheless, patients who develop AKI and severe AKI during VA-ECMO treatment have a higher mortality rate [11]. Research shows that between 2003 and 2014, approximately two-thirds of the 17,942 patients treated with ECMO developed AKI, and one-fifth of those developed dialysis-requiring AKI [12]. Other studies indicate that the overall incidence of severe AKI and AKI requiring renal replacement therapy (RRT) in ECMO patients is 62.8% and 44.9%, respectively. Patients with RRT-requiring AKI during ECMO have a 2.7-fold higher risk of in-hospital mortality than those with AKI not requiring RRT [10]. Patients treated with VA-ECMO are at risk of developing AKI during their ICU stay, and at one year, they may experience a significant decline in glomerular filtration rate [13]. Experiments have shown that within two hours of ECMO initiation, kidney biopsies from pigs revealed leukocyte infiltration, edema, and focal hemorrhage [12]. Therefore, the high prevalence of AKI among patients treated with ECMO is not uncommon. Given the high incidence of AKI, timely prevention could positively impact patient outcomes [8]. Early diagnosis and treatment of AKI are crucial to preventing secondary multi-organ failure and improving outcomes in ECMO-treated patients. Thus, there is an urgent need for a method to diagnose AKI early in patients receiving ECMO.

4. Pathophysiological mechanisms

The development of acute kidney injury (AKI) during ECMO use is associated with various factors, potentially related to both the ECMO treatment and the underlying disease. Studies have shown that patients undergoing ECMO often suffer from severe, life-threatening respiratory and cardiovascular failure, as well as multiple sources of kidney injury, including hypotension, hypoperfusion, anemia, sepsis, and exposure to nephrotoxic agents [14,15], all of which are major causes of AKI [16]. ECMO itself may be a trigger for kidney injury due to non-pulsatile blood perfusion, inflammation caused by exposure to foreign membranes, hemolysis, and hemoglobinuria during ECMO treatment [17].

4.1. Patient factors and critical illness

In the absence of ECMO treatment, the occurrence of AKI can be related to hemodynamic instability, low cardiac output, high intrathoracic pressure, exposure to nephrotoxic drugs, severe hypoxemia, hypercapnia, systemic inflammatory/immune-mediated effects, and neurohormonal dysregulation [18,19].

Cardiogenic shock (CS) and AKI are closely related. During the onset of CS, blood output from the heart significantly decreases, leading to insufficient blood supply to tissues and organs, including the kidneys. The main manifestations include:

(1) Ischemia: CS-induced hypotension and/or low cardiac output significantly reduce renal blood flow, leading to kidney ischemia. In such conditions, renal tubular cells can become damaged and progress to AKI.

(2) Embolism: In CS, slow blood flow or cardiogenic embolism may cause thrombus formation, obstructing renal blood vessels and inducing AKI.

(3) Systemic Inflammatory Response Syndrome (SIRS): After myocardial infarction or cardiac surgery, SIRS can occur, increasing vascular permeability and causing blood leakage from the kidneys, leading

to AKI due to inadequate renal blood supply.

(4) Neurohormonal Activation: CS typically triggers stress responses in the body, such as activation of the sympathetic nervous system and the adrenal renin-angiotensin system, which may increase cardiac output but also cause renal damage, further elevating the risk of AKI.

The occurrence of AKI is also associated with conditions such as heart and/or respiratory failure, the use of vasoactive drugs, sepsis, and ischemia. In heart failure patients, impaired cardiac function, increased intra-abdominal pressure, and renal congestion can reduce renal blood flow and lead to cardiorenal syndrome [20]. AKI can also arise from complications related to other critical illnesses, including bleeding, limb ischemia, infection, coagulation disorders, thrombosis, and neurological events [19-21]. Finally, AKI itself can exacerbate the condition of CS. Renal insufficiency can cause fluid retention, increasing the burden on the heart and further impairing its function. Additionally, renal insufficiency can lead to uremia, directly damaging the myocardium and worsening CS. The mechanisms of AKI development in CS are depicted in **Figure 1**.

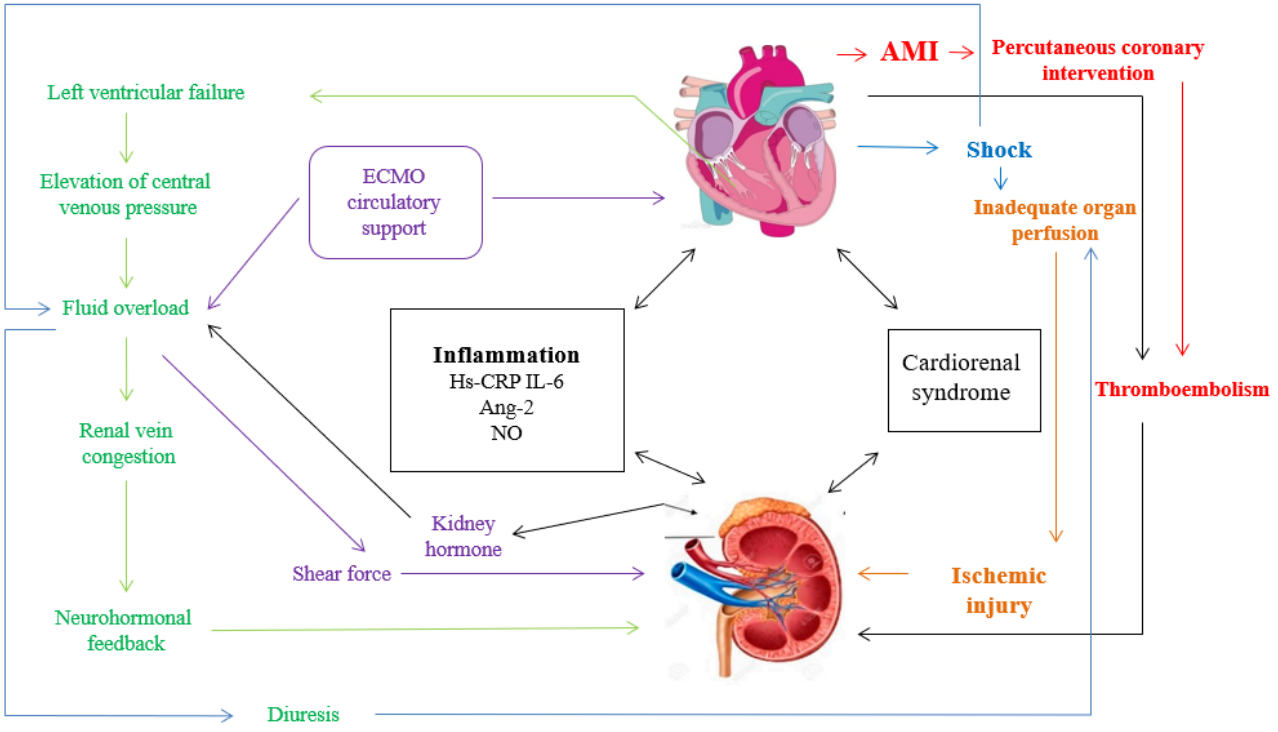

Figure 1. Pathogenesis of Acute Kidney Injury in Acute Myocardial Infarction with Cardiogenic Shock [22]

4.2. ECMO-related factors

(1) Ischemia-reperfusion injury induced by ECMO cannulation: After cannulation, the improvement in oxygenation helps restore the microcirculation of previously hypoxic and under-perfused organs and tissues. The occurrence of AKI is often associated with a certain degree of ischemia-reperfusion injury and the production of reactive oxygen species [23]. During VA-ECMO, the continuous blood flow reduces pulsatility, which may impair renal cortical blood flow and upregulate the renin-angiotensin-aldosterone system, causing systemic vasoconstriction [24]. Finally, although VA-ECMO improves oxygenation and peripheral circulation, limited left ventricular unloading and low ejection fraction can result in left ventricular overdistension and worsening pulmonary edema.

(2) **Inflammatory cascade reaction related to ECMO circuit:** Blood exposure to non-native biological membranes induces the release of inflammatory cytokines, activation of complement and leukocytes, and a hypercoagulable state [18]. Patients with underlying conditions such as infection, hypoxia, and shock, as well as the contact between blood and the ECMO circuit interface lacking endothelial cells, can activate neutrophils, further releasing inflammatory mediators such as tumor necrosis factor-alpha (TNF-α), interleukin (IL)-1β, IL-6, IL-8, and leukocytes, triggering an inflammatory cascade reaction [25-27]. This results in widespread microvascular injury, causing non-septic kidney inflammation, ultimately leading to renal damage.

(3) **ECMO-directly related factors:** These include red blood cell stress and hemolysis. Hemolysis may occur due to the shear stress generated by blood passing through the blood pump, negative circuit pressure, and the contact between blood and the non-biological, non-endothelialized surface of the ECMO membrane. Hemolysis leads to elevated plasma-free hemoglobin, iron release, oxidative stress, and filtered heme, which causes tubular obstruction [28]. Circuit-related factors that contribute to AKI include hemolysis, local ischemia, bleeding, and rhabdomyolysis caused by renal microthrombosis [17,29].

(4) **Cannulation-related complications:** These include improper cannulation positions leading to venous obstruction, cholesterol embolism, aortic dissection, and other complications following cannulation [17,29].

Renal perfusion before and after ECMO treatment plays a decisive role in the occurrence of AKI. Although the kidneys receive about 20% of the cardiac output, with low oxygen extraction in some parts, they are highly susceptible to ischemic injury [30].

5. Factors influencing AKI complications during ECMO

The risk factors for AKI reported during ECMO treatment primarily include advanced age, pre-existing comorbidities (such as cirrhosis), post-cardiac surgery shock as an ECMO indication, delayed ECMO implantation, reduced left ventricular ejection fraction, intraoperative transfusion, elevated lactate levels, increased plasma-free hemoglobin, elevated bilirubin levels, and a high neutrophil-to-lymphocyte ratio [8]. One study [31] indicates that higher pump speeds are associated with hemolysis, destruction of leukocytes and platelets, and complement activation. To prevent AKI related to heme pigment, the pump revolutions per minute (RPM) should be limited to a safe level to avoid excessive negative pressure. Sequential organ failure assessment (SOFA) scores before ECMO, left ventricular ejection fraction before ECMO initiation, and lactate levels 24 hours after ECMO implementation are independent risk factors for early AKI in patients receiving ECMO treatment. This suggests that severe preoperative organ dysfunction, hemodynamic instability due to reduced cardiac output before ECMO initiation, and early microcirculatory hypoxia after ECMO initiation increases the risk of AKI development [30]. Another study [32] shows that a higher extracorporeal life support (ECLS) flow index is associated with an increased likelihood of stage 3 AKI and renal replacement therapy (RRT) incidence, while a lower flow index may not increase the risk of renal injury.

A study by Liao *et al.* [33] found that patients who underwent cardiopulmonary resuscitation before ECMO, those with hyperlactatemia, and the use of high doses of inotropic drugs during ECMO were risk factors for AKI.

Research by Zhao [34] found that hypertension and concurrent infection were also risk factors for ECMO-related AKI. Patients with hypertension have poorer renal reserve function, which makes critically ill

patients more susceptible to AKI. Due to the invasive nature of ECMO and the exposure of blood to the non-endothelialized ECMO circuit surface, the innate immune inflammatory system is extensively activated, increasing the risk of infection in ECMO patients. The systemic immune-inflammatory response caused by infection is the basis for renal pathological changes.

A retrospective cohort study [35] indicated that the use of vasopressin, nursing activity scores, and glomerular filtration rate were factors associated with AKI development in ECMO-treated patients. The factors influencing ECMO-related AKI are summarized in **Table 1**.

Table 1. Factors, mechanisms, markers, and pathophysiology of AKI complications during ECMO therapy

Category	Factors	Mechanism	Markers	Pathophysiology
Patient-related factors	Advanced age Comorbidities (e.g., cirrhosis) Post-cardiac surgery shock Reduced left ventricular ejection fraction Intraoperative transfusion High lactate levels Increased plasma-free hemoglobin Elevated bilirubin High neutrophil-to-lymphocyte ratio Sequential organ failure assessment (SOFA) score Cardiopulmonary resuscitation before ECMO Hypertension history Concurrent infection	Right ventricular failure Reduced cardiac index	Central venous pressure Serum bicarbonate, lactate	Renal venous congestion → neurohormonal feedback → tubular cell injury → inflammation Cardiorenal syndrome Ischemic injury due to inadequate organ perfusion
ECMO-related hemodynamic variables	Delayed ECMO implantation Pump speed Higher ECLS flow index Use of high doses of inotropic drugs	Mechanical circulatory support	NT-ProBNP	Loss of pulse blood flow → shear stress Fluid overload → renal venous congestion Extracorporeal circulation hemolysis
Circuit-related factors	Blood shear force Rhabdomyolysis Hemolysis and oxidative stress Embolism Aortic dissection	Thromboembolism		Cellular damage
Systemic inflammatory response syndrome	Systemic inflammation Renal macrocirculation/microcirculation dysfunction Biocompatibility issues Blood/air/surface interactions Hypercoagulable state	Inflammation	hs-CRP IL-6 Angiopoietin-2 Nitric oxide	Mitogen-activated protein kinase (MAPK) → regeneration of damaged tubular epithelial cells → pathways of regeneration of damaged tubular epithelial cells Capillary leakage

6. Research on biomarkers for ECMO-associated AKI

In recent years, the study of early diagnosis of AKI has developed rapidly, and several new biomarkers for kidney injury have been discovered for AKI assessment, such as serum kidney injury molecule 1 (KIM-1), insulin-like growth factor binding protein 1, and neutrophil gelatinase-associated lipocalin (NGAL) [36-40]. A study by Chinese researchers Li *et al*. [41] demonstrated that serum cystatin C (CysC) and NGAL are effective

parameters for predicting acute/chronic renal failure in patients using VA-ECMO.

In 2019, a study indicated [42] that KIM-1 is a highly sensitive biomarker for acute kidney injury. It was the first to confirm that AKI was caused by VA-ECMO rather than Impella, possibly involving the shedding of the extracellular domain of KIM-1 from renal cortex cells, which increases urinary KIM-1 levels.

In summary, although there is extensive research on biomarkers for AKI, most studies focus on the early diagnosis of AKI caused by other diseases, while there are few reports on early risk prediction of AKI in ECMO-treated patients. Moreover, the measurement of these new biomarkers has not yet been widely applied in clinical trials. Therefore, there is an urgent need for an early risk prediction method for ECMO-associated AKI based on conventional clinical parameters. Chinese researchers have also explored predictive models. Ding *et al.* [43] found that the baseline Model for End-Stage Liver Disease excluding INR (MELD-XI) score before surgery has predictive value for the risk of AKI in patients undergoing VA-ECMO after cardiac surgery. The MELD-XI score can be calculated after VA-ECMO support to assess the patient's AKI risk.

7. Treatment methods for ECMO-associated AKI

The occurrence of AKI during ECMO use is not uncommon in clinical practice, as ECMO itself, due to its inflammatory response, interaction between blood and artificial surfaces, and underlying diseases, may increase the risk of AKI. Currently, the main treatment methods for ECMO-associated AKI include the following:

(1) Early renal replacement therapy (ERRT): ERRT includes Continuous Renal Replacement Therapy (CRRT) and Intermittent Renal Replacement Therapy (IRRT). Both therapies help remove excess fluids and waste products from the body, alleviate symptoms of uremia, improve fluid management, and correct electrolyte and acid-base imbalances [17,44]. In recent years, the development of CRRT devices integrated with ECMO equipment has enabled simultaneous replacement therapy for lung, heart, and kidney functions within the same system [18].

(2) Optimized ECMO management: This includes normalizing hemodynamic status, avoiding hyperventilation and high inflating pressures in the lungs, and maintaining appropriate circulating blood volume to reduce blood damage caused by ECMO flow.

(3) Management of comorbidities and electrolyte balance: This involves optimizing mechanical ventilation settings, monitoring and managing electrolyte disorders, and minimizing drug-induced kidney damage whenever possible.

(4) Blood purification: Techniques such as plasma exchange and high-flux hemodialysis can be used to clear inflammatory mediators, improve microcirculation, and reduce organ damage.

(5) Other treatment methods: These include pharmacological treatments (such as diuretics and vasoactive drugs), maintaining fluid and electrolyte balance, and other supportive measures.

In addition, nursing care and psychological therapy are also important components of actively treating ECMO-associated AKI. Given the physical and mental health impact of ECMO treatment on patients, close cooperation between patients, their families, and the medical team is essential.

8. Conclusion

ECMO has been in clinical use for more than 50 years, and in the past two decades, it has rapidly developed,

providing effective circulatory support, oxygen delivery, and cardiac function support for refractory cardiogenic shock, as well as circulatory replacement for intractable cardiac arrest. Improvements in equipment and consumables have driven clinical progress, and treatment concepts and applications have become more systematic and standardized. The issue of AKI in patients with cardiogenic shock undergoing VA-ECMO has attracted the attention of many experts. Given the high prevalence of AKI, studies have suggested that early initiation of renal replacement therapy (RRT) may improve patient survival rates and reduce the risk of complications. In recent years, tightly integrated CRRT/ECMO systems have been applied in clinical practice, achieving integrated treatment for acute heart and kidney dysfunction. It is also necessary to establish diagnostic criteria for AKI and consider the risk of AKI after ECMO withdrawal. Addressing the issue of VA-ECMO-associated AKI in patients with cardiogenic shock requires collaboration from a multidisciplinary team, including cardiology, emergency medicine, critical care, and nephrology. Further research is needed in the early identification and assessment of AKI, the development of preventive strategies, and kidney protection and recovery.

Disclosure statement

The authors declare no conflict of interest.

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